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St. Bartholomew's Hospital Journal,

APRIL 14th, 1897.

"Æquam memento rebus in arduis
Servare mentem."—*Horace,* Book ii, Ode iii.

Some Chapters on Pneumothorax.

By SAMUEL WEST, M.D.

I. The Physiological Consequences of Perforation of the Lung.

AS soon as the air has gained access to the pleura the elasticity of the lung comes into play and leads to its collapse. Whether the elasticity of the lung is alone sufficient, as is often stated, to produce complete collapse or not is a question which is of more theoretical than practical importance in pneumothorax; for in most cases, in the early stages, the air enters the pleura on inspiration with greater ease than it can escape on expiration, and thus there is added to the forces tending to produce complete collapse of the lung the compression of it during expiration. The result is that in a very short time the lung becomes completely collapsed and airless.

If the lung be free from adhesions it lies shrunken about its root and flattened against the vertebræ. If, however, there be adhesions it will contract in an irregular fashion. If the adhesions are at the apex, as they commonly are, the contraction of the lung takes place in a more upward direction. If the adhesions be in front, along the sternum, the lung may be flattened sideways and lie like a pancake between the sternum and the spine. In such a case as this the heart and mediastinum may be fixed in their usual place, and no displacement of organs occur. When the adhesions are limited the lung in the corresponding part may be drawn out into a long band, which I have seen stretch across the pleura and be some inches in length.

These adhesions and the peculiarities of contraction of the lung to which they lead it is important to bear in mind, as they may explain some of the irregular physical signs occasionally met with.

The effect of the collapse of the lung on the affected side is to render it absolutely useless for the purposes of respiration.

Displacement of organs.—The heart and mediastinum are firmly fixed to the spine behind and but loosely attached to the sternum in front, so that they are capable of considerable displacement, much as a door swings on its hinges. The mediastinum occupies its usual place in the middle line because of the elasticity of the two lungs which balance it on either side. If then the elastic traction of one lung be abolished, as it is in pneumothorax, that of the other side, being unopposed, comes into play and drags the heart and mediastinum over on to the sound side. This displacement is the necessary consequence of pneumothorax if the mediastinum be free to move, and it can only be absent under two conditions—first, when the mediastinum is fixed by adhesions; or secondly, when the other lung has also lost its elasticity or power of retraction.

Although, as Douglas Powell showed, elastic contractility

of the sound lung is of itself sufficient to account for the maximum displacement of the heart and mediastinum, still it is supplemented, at any rate in most cases, by the expiratory compression, as already described.

The effect upon the opposite lung in a case of pneumothorax, therefore, is considerable; for, whereas it formerly filled the whole of that side of the thorax, it now occupies a space so much smaller by the room that the displaced heart and mediastinum take up. This may be roughly estimated at perhaps a quarter. If, for the sake of argument, we assume that the two lungs may be represented in capacity by 5 for the right and 4 for the left we have a reduction in the respiratory capacity in the case of pneumothorax which we may express as follows:—The total respiratory capacity of the two lungs—the whole respiratory capacity of the one and a quarter of the other. In figures this would be—

For right pneumothorax, $9 - (5 + 1) = 3$.

For left pneumothorax, $9 - (4 + 1\frac{1}{4}) = 3\frac{3}{4}$.

Thus the total respiratory capacity of the lungs is reduced considerably more than a half, it may even be as much as two thirds, and this on the assumption that the opposite lung is healthy. Where that lung is also the seat of disease, the reduction in respiratory capacity will be still greater.

The physiological or functional capacity of the lungs is, however, still further reduced for other reasons. The collapse of the lungs as a whole is associated with a proportionate collapse of the vesicles individually, which not only contain a smaller volume of air, but also offer a smaller aërating surface on which the blood-vessels are exposed to the air.

To some extent an attempt is made to compensate for these defects by increase in the depth and number of respiratory movements, but very ineffectually, while the inspiratory forces are greatly diminished by the fact that only one side of the diaphragm has any effective action at all.

These defects are grave enough, but there is still another, which is perhaps still more important than either, viz. the sudden embarrassment of the circulation which pneumothorax causes.

In the first place, the one lung left is suddenly called upon to do the duty of two. The right heart goes on attempting to pump its usual amount of blood out, but there is only one lung for it to pass into. Unless the blood can pass through the lungs with at least twice the normal rapidity (and that is clearly impossible) the blood must accumulate in the lungs; the vessels will become distended and the lungs congested. As the result of this, the free exchange of gases in the vesicles of the lung between the blood and the air is impaired, and thus the passage of the blood through the lung capillaries is rendered more difficult than before. The heart endeavours to overcome the obstruction by beating more rapidly and more forcibly, but soon it fails, and then the difficulties of the circulation are still further increased, and the already failing heart becomes still more distended.

As long as the remaining lung and the heart are able to cope with the extra work thus thrown upon them, the lung will yield no physical signs; but as soon as the work becomes too much for them, the congestion of the lung will show itself in the usual way with the signs of bronchitis and sometimes with hæmoptysis, while the failure of the right heart betrays itself by increasing dilatation, irregularity, and feebleness of action.

Thus the end may come in two ways, either from congestion of the lungs or from over-distension of the heart. This is the condition appropriately named by Wintrich "*Insuffisance aiguë du poumon.*"

In the causation of the urgent symptoms time is a very important factor. It is not so much that one lung is helpless and the other has to do double duty, but that the change is so sudden that neither lungs nor heart have had time to adjust themselves to the altered conditions. Although in pleuritic effusion one lung may be as completely collapsed and useless and the displacement of organs to the opposite side may be as great as in pneumothorax, still such urgent symptoms are rarely seen. The difference is due to the fact that in cases of pleuritic effusion the changes have been more or less gradual, or at any rate not nearly so sudden. At the same time, even in pneumothorax, when the patient does not succumb at once, if the heart and the lungs be healthy they may adjust themselves to the altered conditions and the urgency of the symptoms then gradually subside.

The great danger of pneumothorax is during the first hour or so after its occurrence, and the immediate prognosis improves with every hour that life is prolonged.

The urgency of the symptoms of pneumothorax depends, therefore, principally upon the suddenness of the change in the respiratory conditions, and next upon the strength of the heart and the condition of the opposite lung.

The maximum change is met with in cases where the previous health has been good and where the lungs have not been obviously diseased. Accordingly, the most intense symptoms in pneumothorax are met with in those who have been previously apparently in good health; while, on the other hand, in some cases of well-marked phthisis the symptoms may be so slight that pneumothorax may be overlooked. This apparent paradox is intelligible if it is looked at from the point of view of the amount of change in the respiratory conditions which is produced in the two cases. Thus, where the patient has been in apparently good health previously, and the lungs are but little diseased, the displacement of organs, and the consequent collapse of the opposite lung, reach their maximum, while the congestion of the opposite lung is extreme, the patient being full-blooded, and all the nutritive processes in full activity. On the other hand, if the patient has been ill for some time, as,

for example, in most cases of phthisis, the amount of blood in the body, and the consequent congestion of the opposite lung, is much less; moreover, one lung has probably for a long time been doing little work, and it may really make but little difference in the respiratory conditions, whether the one lung is useless on account of phthisis, or because it is collapsed as the result of pneumothorax. On the other hand, if the lung on the opposite side be also extensively diseased, the reduction in respiratory capacity may be so great as to be incompatible with life. Yet it is extraordinary how little lung seems necessary in some cases for mere existence, so that even in advanced cases of phthisis pneumothorax sometimes produces hardly any symptoms.

Other things being equal, the strength of the heart (which also depends greatly upon the general strength of the patient) will largely influence the power the heart may have of dealing with the altered conditions when the symptoms are urgent.

So we find in some cases of advanced phthisis the occurrence of pneumothorax may lead, not to the ordinary symptoms of pneumothorax, but to cardiac syncope, which may end in sudden death, or a long collapse, from which the patient may never rally.

Megrim cured by Abstinence from Meat (Haig's Megrim).

By W. P. Herringham, M.D., F.R.C.P.

THE following cases are specimens of a class of sick headache that well repays attention because it can be cured.

Benj. C. A—, a male Jew æt. 12, has had headaches ever since early childhood. They have gradually become more frequent, and now occur twice a week. They are general, and not one-sided. They usually come on at midday. He becomes very pale, has to lie down, vomits, then goes to sleep and wakes up better.

He has already been treated with spectacles by an oculist, but without effect. His diet was carefully examined; it appeared to be thoroughly wholesome. No fault could be found with it on ordinary principles. I therefore advised to entirely leave off butcher's meat, and his mother was sensible enough to carry out the treatment. During the next fortnight he had but one headache instead of four, and in the next month one only instead of eight. I gave him no medicine.

Ethel W—, æt. 20, a healthy-looking girl with a fresh colour, has had megrim ever since she was seven years old. The headache used to come once in six weeks, but has gradually become more frequent, and now seizes her once a fortnight. It begins with epigastric pain and nausea; sudden headache over one eye follows, and hemianopia on the same side. She becomes a livid white, has repeated vomiting, and her fingers become numb. She goes to bed, and is better next morning.

I at first made her take a tea made of guarana when she felt the attack impending, and for a month this kept the headache off. But after that she had a whole week of indigestion, vomiting, and pain in the left temple, and an attack of vertigo and hemianopia to end it.

The eyesight and fundus were quite natural.

After this I persuaded her to give up butcher's meat, and with very happy results. I saw her from time to time during the next few months, and she almost entirely lost the headache.

In neither of these cases was there any affection of the teeth, nor any of the ordinary symptoms of indigestion between the attacks of megrim. Nor could I trace in the family history of either patient gout, rheumatism, or any nervous disease.

This treatment of megrim by abstinence from butcher's meat, and if necessary from other articles of diet, was introduced by Dr. Haig several years ago. He supported it on a theory which I believe to be entirely unfounded, and which has not been confirmed, that the symptoms were due to the circulation in the blood of the salts of uric acid, and other consequences of this. But be the theory right or wrong, I am sure that for a certain set of cases the treatment is successful. I think it worth while to call attention to it, because I do not find it mentioned in ordinary medical writings with the respect it deserves.

These migrainous symptoms are not alone. There are a considerable number of people, both men and women, usually of a nervous temperament, and very often, like patients with megrim, of powers much above the average, who constantly complain not of pain, but of discomfort in the head. They have a sense of fulness, and even of "swimming" in the head; they are unable to do mental work after luncheon, and they complain of a number of little things which one is very much inclined to treat with contempt. It is well worth while in such cases to try what abstinence from red meat will do for them. Most of them have already learnt to abstain from beer or wine, but if not they must be deprived of these things too. It is not the primary digestion that is at fault. They do not usually have pain or flatulence, or a furred tongue. But, for some as yet undiscovered reason, while fish, fowl, milk, and the vegetable proteids are assimilated well, red meat is not.

I have known several of these people entirely relieved from their symptoms by this treatment, and I have even known a person so treated recover the power to drink wine, which used at one time always to disagree.

I should say, to avoid disappointment, that cure is not sudden in these cases, though it is rapid. An occasional recurrence at first of the headache or of the sense of discomfort must be expected.

Pathological Jottings.

By A. A. KANTHACK, M.A., M.D., Deputy for the Professor of Pathology, Cambridge University.

V. ACUTE INFLAMMATION.

TO give a definition of Acute Inflammation is almost an impossibility. Numerous attempts have been made from time to time, but it must be confessed that none of them have been altogether successful. We need not go back to Virchow, because his views on this subject belong to the past and have been forgotten by a precocious generation; moreover few people now-a-days read his works, although they are full of treasures. More recent definitions have been supplied, notably by M. Metschnikoff and Professor Adami. The former, studying inflammation chiefly in its relation to bacteria, builds up a theory upon phagocytosis as a foundation. He observes the changes in mammals, frogs, crustaceans, amœbæ, and finds that phagocytosis is the one phenomenon which is seen in all animals in the struggle against micro-organisms, and therefore argues that it is the *primum movens* of inflammation. Such reasoning is unsound, because the analogy is incomplete, and it is highly questionable whether we can analyse such a process as inflammation, which is extremely complex, and, so far as it is known to us in its true form, occurs in extremely complex animals by going back to such simple animal forms as daphnia and amœba. We might as well try to study mental activity in man and the higher animals by examining an amœba. A process occurring in an animal possessed of highly differentiated nervous and vascular systems, the tissues of which, moreover, are highly complex, cannot be compared to any process observed in an animal the structure of which is very simple, or is represented by a single cell. Structure can be traced more easily and convincingly by the evolutionist; it is, however, a different matter when we are dealing with functions, and what are vaguely called processes or reactions. In defining inflammation, or in describing it, we must restrict ourselves to classes of animals homologous in structure, and known to react by what we recognise as inflammation.

Professor Adami defines inflammation as *the local attempt at repair after an injury actual or referred.* This definition covers too much ground; it includes phenomena which no histologist would or could regard as inflammatory. If we take Professor Adami literally, the constant renewal of the cuticle would be inflammation. An injury—as, for instance, superficial epithelial lesions—may be repaired without what is generally recognised as inflammation, *i.e.* by regeneration and *direct* repair. Some low forms of animal life are capable of regenerating any part or parts of their body. Regeneration is not the same as repair by inflammation, or *indirect* repair. There is no valid reason of extending the meaning of a term so as to make it answer the requirements of a definition. We know inflammation by its phenomena and its appearances, and by the changes in the tissues, and unless all these are present are we justified to speak of inflammation? Here, as elsewhere, we must be strict in our terminology. A certain process or an attempt at repair in a low form of animal may in some or in many of its phenomena resemble inflammation, yet without being inflammation; just as a cutaneous papilloma may resemble a squamous-celled carcinoma without being a carcinoma. It is unsafe to be too philosophical; inflammation is a complex process which occurs in complex tissues; our criterion of inflammation is what we know to occur in such tissues, and we are not justified in calling inflammation what does not agree with our observations on animals which show the recognised reactions of inflammation in their tissues.

We must start from this, and we shall find that acute inflammation is a reaction of mixed tissues which occurs only in man and other vascular animals; it is a uniform process, varying, no doubt, in its different types, but in degree only, not in kind; there is a uniformity in the pathogenesis, progress, and morphological attributes of acute inflammatory conditions which is so striking that we must demand, that nothing be called inflammation, unless it presents all the essential phenomena which the study of disease in man and other vascular animals has revealed to us. To select phagocytosis, or chemiotaxis, or new formation and repair as essentials, and making them the corner-stones of theories of inflammation which are rendered more attractive by an appeal to comparative anatomy and evolution, is unjustifiable; phagocytosis, chemiotaxis, and proliferation are concomitant, or it may be constant, phenomena of acute inflammation, and each one of them by itself can readily be traced back from the highest to the lowest form of animal; but surely it is not sound reasoning to evolve the whole process of inflammation from one or two of its phenomena, especially when such phenomena are extremely primitive protoplasmic properties. Evolution may teach us how a property or a character has been acquired, it may tell us something of the phylogenetic origin of an organ or a process; but it does not pretend to assert that a complex process in a higher animal type, which perfectly or imperfectly can be traced back to some property or function in a low animal type, is identical with this property or function. In appealing to evolution we constantly lose the thread, and find many gaps which we cannot fill up. Inflammation, as it is known to the human pathologist, occurs only in certain higher animals. *Without a blood-vascular system inflammation is impossible.* This is the demarcation line, and therefore we cannot go back to *avascular* animals for an explanation. A vascular animal reacts to a certain stimulus by inflammation, while the same stimulus in an avascular

animal may produce some phenomenon which also occurs in inflammation, but itself is not inflammation.

Taking acute inflammation as we find it, and analysing the process, we come to the conclusion that (1) without blood-vessels there is no inflammation, and (2) it is a reaction of vascular connective tissue or of connective tissue, itself perhaps avascular, but in immediate and close relation with the vascular system. Inflammation of epithelium does not exist, and nobody has ever yet observed true inflammation in *really* avascular connective tissue, such as cartilage. Those who believe in the existence of acute inflammation in avascular tissue invariably quote experiments made on the cornea Now no doubt the cornea is anatomically an avascular tissue, but it is in extremely close connection with the circulation by means of the vessels at its periphery and its numerous lymph channels. When we produce so slight a lesion of the cornea that nothing more follows than a limited destruction and proliferation of the corneal corpuscles we are not dealing with inflammation, but simply with direct repair: any tissue that has life left after an injury or a loss of substance will at once repair itself or regenerate. During lactation the cells lining the mammary alveoli are constantly losing part of their substance, but they immediately repair the loss. Is that inflammation? As soon as the injury is severe enough, so as to transmit its influence to the vessels around the cornea, all the changes of inflammation become evident. The cornea, therefore, is a connective tissue which is subject to inflammation if the stimulus be adequate; if not, repair occurs without inflammation. We know that an irritant may produce different effects according to its intensity and the method of application; it may produce a slight injury, easily and directly repaired, or a serious injury, followed by immediate death or slow necrosis, or followed by secondary inflammation, or it may produce an acute primary inflammation. Now because the irritant is one and the same substance, and has remained unchanged, while experimenting on a graduated series of animals, we have no right to assert that the effect of its action is one and the same process, whatever the tissue and whatever the animal. No doubt, if we compare the different effects produced, there is a gradual transition from one to the other, but so there is from an innocent to a malignant growth.

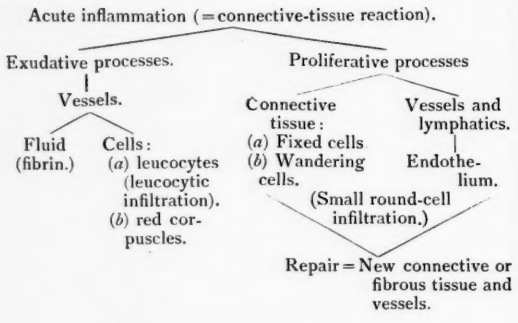
A few vibrations of the mysterious ether around us will produce the subjective sensation of sound, while innumerable vibrations of the same ether will produce one of light, yet sound is not light. The cornea experiments, therefore, cannot be used as arguments against the view, originally supported by Cohnheim, namely, that inflammation can occur only in vascular tissues, because as soon as we produce changes which every one would recognise as inflammation, the vessels around the cornea and the tissue in which they lie have reacted, being, so to speak, drawn into the zone of irritation through the innumerable lymph channels. What is observed in some avascular animals may be considered to be or to effect whatever inflammation is or does in a vascular animal, but it is therefore not inflammation.

Inflammation, in my opinion, then, must be regarded as a series of changes occurring only in vascular or vascularisable connective tissue, or in connective tissue which is in easy communication with the surrounding blood-vessels. Starting from this point, we may pass over the causes of acute inflammation by merely mentioning that traumatic, chemical, or physical irritants, including foreign bodies and micro-organisms, are capable of reacting on the connective tissue in such a manner as to produce the phenomena and appearances of inflammation. These irritants must possess a certain *relative* intensity, otherwise inflammation may not set in, or if the intensity is too great necrosis may be the result. The intensity will, of course, vary with the general or local tissue resistance of the individual. Thus in renal disease or *morbus cordis* or venous engorgement, or in certain nervous lesions, inflammation is readily produced by conditions which are incapable of producing it in the healthy man. Necrosis, it must be remembered, may act as an irritant and be followed by inflammation; dead tissue is either absorbed, encapsuled, or cast off, or it is replaced by regeneration, and the absorption or casting off is often or even generally accompanied or effected by inflammation. No doubt the products of necrosis act as an inflammatory irritant. It is difficult to separate necrosis from inflammation; the former may lead to the latter, and *vice versâ;* some cellular necrosis almost always occurs with acute inflammation. Necrosis or necrobiosis, however, is not always followed by acute inflammation, but may be the forerunner of fibrosis or so-called chronic inflammation, as I have stated elsewhere (*vide* "Chronic Inflammation").

Passing on now to the phenomena of acute inflammation, we may distinguish two main processes, (*a*) exudative and (*b*) proliferative. The *exudative processes* are, speaking generally, most evident during the earlier stages, and are concerned especially with the vessels. Fluid and cells may pass through the vessel wall, the fluid being coagulable lymph, which may or may not coagulate, the cells being leucocytes and red corpuscles. The *proliferative processes* are observed in the connective tissue and endothelium and in the vessels. The fixed and wandering connective-tissue cells multiply, and so do the endothelial cells lying in the lymph spaces and lining the capillary and lymphatic walls. These proliferative changes are best seen in the later stages of acute inflammation. It appears, therefore, that in the earlier stages, if many cells are present in the inflamed area, they are mainly leucocytes, *i. e.* we have a *leucocytic infiltration;* while in the later stages the cells are mostly derived from the connective tissue or endothelium, forming the so-called *small round-cell infiltration*. The products of

proliferation, if repair follows, become converted into fresh connective or fibrous tissue, but when this occurs inflammation is at an end. Although we have said that in the earlier stages of inflammation the exudative changes are especially evident, it must not be imagined that proliferative changes are absent altogether during that early period; in fact, at the seat of irritation itself the proliferative changes may be the first to show themselves. In a general way it may be said that the more vascular a part is the more evident are the exudative changes during the earlier stages; and conversely, the less vascular a part is the more marked are the proliferative changes during the earliest stages. But this much is certain, that in any process which is recognised as inflammation there are always both exudative and proliferative changes.

We shall now discuss in more detail the different phenomena of acute inflammation, and begin with the exudative processes. This, however, must be left for a future occasion.



Cases of Acute Poisoning

by Atropine, Cocaine, Carbolic Acid, Potassium Bichromate, and Aconite.

By J. D. RAWLINGS, M.B.Lond., M.R.C.S., late House Physician, St. Bartholomew's Hospital, &c.

CASES of emergency, either medical or surgical, have always an interest peculiar to themselves; and as any acute poisoning is an emergency *par excellence*, I offer the notes of the following cases which, among others, have come in my way during the last few years.

Atropine.

F. T—, æt. 6, brought to St. Bartholomew's Hospital on September 10th, 1895, by his father, who complained that for the last three hours the boy had been "off his head." The patient's eyes were under treatment, and his mother was dropping a solution of atropine into them at intervals. Three days ago, when this was done the boy "seemed silly" for an hour or two afterwards. At 6 p.m. on September 10th the drops were again put into his eyes, and at 9 p.m. he again became silly and grew worse. The father was quite certain that the child had not swallowed any of the solution. When seen at midnight the patient was wildly delirious, seeing imaginary objects around him, and expressing extravagant surprise or amusement at them. He was not frightened at any of his hallucinations, appearing, on the contrary, highly exhilarated. When interfered with he struggled violently and cried out. Pupils widely dilated, and not reacting to light. The boy did not look ill. There was no rash, nor did any appear subsequently. There was no evidence that his skin or throat were causing him any discomfort. It seemed only prudent, in spite of the history, to give an emetic, and as the boy refused to drink anything a hypodermic injection of apomorphine gr. $\frac{1}{20}$ was given, which was followed in a minute and a half by copious vomiting with little or no nausea. As the delirium and vomiting continued a hypodermic injection of morphia gr. $\frac{1}{10}$ was given, which was followed by several hours' sleep. In the morning the child was still a little inclined to talk nonsense, but was quite quiet, and was sent home with his parents.

The case was not a severe one, and its chief interest lies in the mode in which the poisoning occurred. It is fortunately not common for the instillation of atropine drops into a child's eyes to be followed by toxic effects, but they may occur apart from any carelessness in the use of the drug, as most ophthalmic surgeons have found. The risk may be reduced to a minimum by compressing the lachrymal sac with the finger for a minute or so after putting the drops into the eye, or by using ointment instead of a watery solution. Where atropine has to be applied at home by the child's parents ointment has the additional advantage over drops, that it is less attractive as an article of diet.

Cocaine.

R. P—, æt. 25, brought to St. Bartholomew's Hospital September 13th, 1895, for convulsions. About a year previous to the present attack patient acquired the habit of taking cocaine by the mouth, in the hope of curing himself of a craving for spirits, which he had already developed. He has cured himself of drinking, but in the course of the past year has had several attacks like the present one. At 5 p.m. on the day of admission patient took six grains of hydrochlorate of cocaine by the mouth. At 7 p.m. he began to have convulsions; he was said by the policeman who brought him to have been unconscious and struggling violently when found. When seen in the surgery at 7.30 he was having violent convulsions suggestive of epilepsy, but when his supra-orbital nerve was irritated he talked clearly and coherently, and said that he had been taking cocaine. The pulse was then very rapid. He had not micturated nor defæcated during the fits. At 9 p.m. his face and body were wriggling all over, the movements being indis-

tinguishable from those of severe chorea. The pupils were dilated, but reacted to light. Knee-jerks perhaps slightly increased. When told to put his tongue in again after protrusion it flew back with the spring-like action seen in chorea. There was no rash then or subsequently. Pulse 108, irregular in time and volume, occasionally intermitting; heart otherwise normal. Respiration natural. Patient had never had rheumatism or chorea, nor any attack at all like the present one till after he had begun taking cocaine. He was quite well the next day, and was discharged.

Although convulsions are a common feature in cocaine poisoning it is quite unusual for the movements to closely simulate chorea. Convulsions caused by cocaine are of cerebral origin, since they cease on division of the cord (Brunton). Chorea is due apparently to either a neurosis or an altered blood state acting on the cerebral cortex (Gowers). The altered blood state which leads to chorea is, generally speaking, associated with the rheumatic poison, whatever that may be. Is it not conceivable that cocaine may in exceptional cases have the same effect on the cerebral cortex as the rheumatic poison, thus causing not a neurotic or rheumatic chorea, but a condition which might with propriety be called "cocaine chorea," but which lasts only until the cocaine is excreted by the kidneys? This is a mere speculation, not an opinion. The case is a fair example of the efficacy of treating the alcohol or morphia habit by substituting another drug for the alcohol or morphia.

Carbolic Acid.

F. F—, æt. 39, music publisher, brought to the Royal Free Hospital at 10 p.m., May 22nd, 1894 Patient had been drunk during the evening, and had drunk about two ounces of strong commercial carbolic acid. He was seen at the hospital a quarter of an hour after taking the poison. He was then comatose with stertorous breathing. Corneal reflex absent. The skin was cold, hands and face livid; about the face and chest were patches whitened with carbolic acid. Lips, tongue, and palate whitened; uvula very œdematous. Breath smelled strongly of carbolic acid. Pulse 120, good volume, regular. Respiration 24. Patient was wrapped in blankets, and hot water tins were applied to feet and legs. The stomach was washed out twenty or thirty times until the washings were odourless and clear. Eight ounces of olive oil was then put into the stomach. The pulse was now smaller in volume, and irregular. A drachm of brandy was injected into the chest wall. The corneal reflex now returned, though the patient remained unconscious. At 11.30 p.m. he was removed to the ward. Temperature 95° F. Patient now began to recover consciousness, but for some hours after admission could not breathe without having his tongue held forward. Temperature at 6 a.m. 97·6°; at 10 a.m., 100·6°. During May 23rd patient was quite conscious and rational, complained of thirst, but not of pain in stomach or elsewhere; was fed by rectum. Slight general rhonchus, well-marked carboluria and a little albumen in the first specimen of urine passed after admission. May 24th.—Seemed fairly well. Pulse 136, good volume and tension. Respiration 44. Temperature 99° F. Considerable quantity of muco-purulent sputum. Crepitations at both bases behind, and signs of consolidation at the right. The patient was removed in the afternoon by his friends, who had their own reasons for wishing to take him home. I heard subsequently that patient died of double pneumonia on May 29th, *i.e.* seven days after taking the poison.

The immediate danger of carbolic acid poisoning is from paralysis of the nerve centres, evidenced by coma and failure of pulse and respiration. This is not due to shock caused by corrosion of the alimentary tract, for the latter is rendered anæsthetic whenever the phenol touches it; and, moreover, the nervous symptoms of carbolic acid poisoning are the same when absorption takes place by the skin. The general condition of the patient then, though superficially similar, is quite different in origin to that caused by a corrosive mineral acid, such as sulphuric, in which the condition is one of shock, caused by a sudden destruction of mucous membrane richly supplied with nerves. There is a very practical difference, also, between the local conditions in the two cases, for though carbolic acid is often spoken of as a corrosive poison, and we are warned to be careful about passing the tube, the œsophagus and stomach present a very different appearance in phenol poisoning from what they do in a sulphuric acid case. In the latter the walls are charred and rotten, so that it may be the easiest thing to poke one's finger through them. In the former the inside of the stomach and gullet present the appearance of having been tanned, and it is, if anything, rather more difficult to tear their walls than those of healthy organs. Since no safe antidote to carbolic acid is known, it is as important to empty the stomach as in alcohol or opium poisoning. Emetics are useless because the stomach is anæsthetised, and will not react; or, to quote Dr. F. W. Andrewes, when making the post-mortem on a case which had been treated with emetics, "one might as well pour mustard and water into a stone jug, and expect that to vomit." Even apomorphine does not always act in these cases, and if it did it would scarcely be a suitable drug to give to a patient already collapsed. The only thing to be done, then, is to wash out the stomach. There will probably be more difficulty in passing a tube than in most cases, because the mucous membrane of the pharynx and œsophagus is swollen and hard: it may, indeed, be impossible to pass the soft tube at all, but in my opinion it is safe not only to use the stiff tube of the stomach-pump if necessary, but to use as much force as is necessary to make it pass, assuming, of course, that the operator is familiar with the use of the stomach-pump, and can pass it easily in ordinary cases.

It is well to bear in mind that even when the patient recovers from the immediate effect of the poison he is still liable to bronchitis or pneumonia.

Potassium Bichromate.

J. C—, æt. 20, furniture polisher, brought to St. Bartholomew's Hospital at midnight, August 12th, 1895. Patient's condition did not admit of his giving a very reliable or accurate history, but he was understood to say that he had taken some "chromate of potash" about an hour before, but did not know the quantity. He was found in a railway carriage and brought to the hospital on a stretcher by the police. When seen at midnight, patient was lying on a couch grunting with every expiration. His general condition suggested simply that he was in some discomfort, and was making the most of it. Face flushed; no staining about lips, tongue, or face; abdomen natural, no pain or tenderness; some vomited matter brought by the police showed nothing characteristic; it was dark brown and grumous. Extremities warm. Pupils rather contracted, reacted to light. Patient answered questions but spoke indistinctly. Pulse slow, regular, good volume and tension. On account of the history, and the fact that a packet of crystals of potassium bichromate was found in his pocket, the stomach was washed out. The first washing contained a quantity of food, and there was nothing unusual in its appearance; the next was tinged yellow, but quite clear. When the washing was complete, about two ounces of magnesium carbonate was put into the stomach. No further treatment was adopted for half an hour, at the end of which time the patient was unconscious, slightly cyanosed, pulse of smaller volume and lower tension than before, and the extremities were becoming cold. He was removed to the ward at 1 a.m., where he was surrounded by hot water tins, and enemata of brandy and essence and hypodermic injections of strychnine were given every two hours. The patient gradually sank, and died at 6 a.m. *Post mortem* no lesions were found to account for death. There was no staining, inflammation, or corrosion of the alimentary tract.

Bichromate of potash is one of the irritant poisons,—that is, a poisonous dose causes a sense of burning in the mouth, throat, and stomach; severe pain, vomiting and purging, with collapse. In this case, however, there were no signs of irritant poisoning, nor were any found after death. In fact, on this occasion bichromate acted not as an irritant, but as a neurotic poison very similar to carbolic acid, paralysing the nervous centres and causing death by heart failure. This case, as first seen, was certainly calculated to deceive: a lovesick youth had taken an unknown quantity of irritant poison, and was very sorry for himself, but had no signs of irritant poisoning. What more natural than to wash out his stomach and send him home? It is interesting to note that potassium bichromate in doses of gr. $\frac{1}{10}$ sometimes relieves gastric pain when such drugs as bismuth fail to do so.

Aconite.

B. B—, æt. 44, chemist's assistant. On December 5th, 1894, patient ate too much pork for dinner, and at 3 p.m. drank half an ounce of what he believed to be Tinct. Aurantii, but soon found to be Tinct. Aconiti. At 3.30 p.m. he took half an ounce of ipecacuanha wine, and vomited copiously at 3.45 p.m. Immediately after drinking the tincture he noticed that his tongue tingled very much, but he attributed this to the spirit. In about twenty minutes his limbs seemed to become rigid, and the muscles contracted spasmodically. He then became giddy, and his speech thick; he felt numb all over. A "suffocating feeling came over him," and he developed a craving for fresh air; he accordingly went for a short walk, and while out vomited copiously three quarters of an hour after taking the poison. After vomiting he sat down, and for a time did not know where he was; when he partially recovered from this condition he was unable to see anything, and felt very faint. When the sensation of numbness ceased he had "pins and needles" all over him. The above history was obtained from the patient himself after recovery.

The case was first seen in the Casualty Department at 6 p.m., when the patient was faint, face pale, extremities cold and blue. Breathing unobstructed, about twenty per minute, and deep. Pulse 40; irregular, very small volume, very low tension. Heart-sounds scarcely audible at apex. Patient was quite conscious, and answered questions intelligently. He was wrapped in blankets, and had a hot water tin put to his feet. The stomach was washed out three times, half an ounce of brandy was given by the tube, Liquor Strychninæ ℥iij and Tinct. Digitalis ℥v were given hypodermically. After this the pulse improved, becoming fuller in volume and about 60, but the alternate beats were very weak and the corresponding heart-sounds almost inaudible. In about fifteen minutes' time another hypodermic of strychnine and digitalis was given. Patient was then carried up to the ward, where he was given some more brandy and a purge. Tinct. Digitalis ℥viiss out of Haust. Ætheris Ammoniatus was given every four hours. At 8 p.m the pulse was 64, quite regular, good volume, somewhat low tension. No objective signs of sensory disturbance; but patient said he felt some tingling and numbness in his hands till the following morning. The day after admission he felt quite well; the pulse was 80, regular, good volume and tension.

The symptoms in this case were typical. A very large dose (half an ounce of the tincture) was taken and retained for three quarters of an hour. Digitalis is an invaluable antidote in aconite poisoning when there is heart failure, as there usually is; it should, of course, be given hypodermically. One of the leading text-books on toxicology does not mention this point in the treatment of aconite poisoning.

Vaccination and its Results.

*A Paper read before the Abernethian Society on February 18th, 1897,**

By F. W. Andrewes, M.D., F.R.C.P., Lecturer on Pathology, and Pathologist to the Hospital.

LAST summer Dr. Gee chose for the subject of his address before this Society "The Conflict of Medicine with the Smallpox." In the admirable historical review of the subject which he gave he purposely abstained from entering into the question of vaccination in its controversial aspect. I have had the boldness to select the subject for my paper this evening, partly for the reason that he did not touch upon it, partly because the long-expected final report of the Vaccination Commission has of late appeared, and with it, in the minority report of the Dissentient Commissioners, what may be taken as the very best case which can be made out against vaccination; partly, again, because we have recently had, in the Gloucester epidemic, a striking example of the results which may arise from a wholesale neglect of vaccination. But, above all, I have chosen this subject because I do not think that sufficient intelligent attention is paid to it in medical education. You probably all believe in vaccination in a general sort of way, because that is the atmosphere in which you have been brought up. Yet how many of you are capable of maintaining your belief against a clever and disputatious anti-vaccinationist with a taste for dialectics and a keen nose for statistical fallacies? There is too much tendency amongst us to regard vaccination as a sort of article of religion, to question which is a heresy to be ignored and despised. The matter is a much more complex and difficult one than might at first sight be supposed; and if to-night I can succeed in setting clearly before you some of the arguments for and against the practice, so that you may have a secure basis for the faith that is in you, I shall have no reason to regret my choice of a subject. The opponents of vaccination spare neither labour nor expense in the diffusion of their views; it is your duty, if you are sincere believers in vaccination, not to pass by their propaganda in contemptuous silence, but to arise and publish your gospel to the ignorant with an intelligence and conviction born of accurate knowledge, and with a full appreciation of the arguments which can be raised against you.

The difficulty of the subject depends largely on the fact that the strongest arguments in favour of vaccination are nowadays based on statistical evidence, in the interpretation of which much care is necessary. Vaccination is probably only one of the factors which has influenced the decline of smallpox, and the problem is to disentangle and justly appraise the share which it has borne. The essential questions at issue are practically these:

(1) What is vaccinia?

(2) What degree of immunity does it confer against variola?

(3) What disadvantages or dangers attach to the practice of vaccination?

(4) What ethical right has the majority, in this particular case, to enforce its views upon the minority?

It is with the first two questions only that I shall mainly endeavour to deal in the time at my disposal this evening.

I. *What is vaccinia?* and what is its exact relation to variola? The answer given by modern pathology practically confirms Jenner's original assumption. It is variola, modified and attenuated in virulence by transmission through a relatively insusceptible animal. This is a sweeping assertion. Let us see if it can be justified. The arguments are as follows.

(1) There is an *a priori* argument to which weight, though not the greatest weight, must be attached. We have incontrovertible evidence that vaccination does, for a term of years at least, protect against smallpox. I do not think this is disputed by any person competent to judge the facts. Within the past few years we have begun to know something definite and tangible about the essential nature of protection and immunity in specific diseases. If vaccinia were not a modified form of variola, the fact that it protects against it would be unique and unexplained—out of harmony with an otherwise consistent body of pathological facts. It is, of course, not impossible that a temporary antagonism may exist between two distinct specific diseases, but I know of no facts lending colour to the supposition that an attack of one specific disease can confer an immunity against another and different specific disease stretching over a term of years. One may hold very liberal views as to the limits of specificity amongst micro-organisms; yet smallpox is at least specific among diseases in the same sense and degree as an oak amongst plants, and there are reasons for comparing vaccinia, in this connection, with a dwarfed and stunted oak grown in uncongenial soil. Recent advances in modern pathology and serum therapeutics indicate that immunity is, in a sense, specific too. We are now familiar enough with the fact that many pathogenic micro-organisms can be attenuated in virulence by transmission through relatively insusceptible animals. The important fact about vaccinia is that the mitigation of virulence remains permanent; this, however, is by no means without parallel amongst other pathogenic organisms. Regarded as a modified variola, vaccinia takes its place naturally in a consistent body of pathological doctrine; the alternative supposition places it out of harmony with all that we know about other specific diseases. This is merely argument from analogy, and may be deemed an unsafe guide. Let us now turn to facts.

(2) If vaccinia is only modified variola, intermediate stages should be traceable. Such undoubtedly exist. In its characteristic form vaccinia differs from smallpox in being a disease localised at the seat of inoculation. The inoculated form of smallpox was typically followed, after the ordinary incubation period of about twelve days, by a generalised eruption. Now such a generalised eruption does occasionally follow vaccination; vaccinia rashes are usually erythematous, but may be papular, vesicular, or even pustular. A secondary crop of vesicles may develop around the points of inoculation, or they may be generalised over the whole body. Such cases, which are rare, may, I think, be justly regarded as intermediate forms between vaccinia and variola, though to be classed with the former and not with the latter. It must be remembered that in the last century some of the inoculators had been able to reduce inoculated smallpox to a localised affection without any generalised eruption; this, however, only occurred in exceptionally favorable cases.

(3) A much more convincing proof of the identity of variola and vaccinia is that the latter can be and has repeatedly been produced from the former. This was done nearly sixty years ago by Ceely and Badcock, who each succeeded in producing vaccinia in cows by inoculating them with matter from cases of human smallpox; the lymph thus produced was widely used for human vaccination. It must be confessed, however, that some doubt exists as to these results, and the majority of attempts seem to have failed. In some instances smallpox, and not vaccinia, resulted when the lymph was inoculated upon the human subject; the explanation being that the virus was not sufficiently attenuated by a single transmission through the cow. Twice at least in recent years has the attempt been successful. In 1885 Simpson succeeded in producing a vesicle resembling cow-pox on a cow's teat by inoculation with smallpox lymph. From this cow Cory and Shirley Murphy inoculated a calf. From the calf a child was inoculated; typical vaccinia was produced. In all, eighty-nine calves and 1247 children were inoculated from this strain of lymph; 98·3 per cent. of the inoculations were successful, and nothing but vaccinia was ever produced. In no case was there the slightest trace of a general eruption.

Within the last few years Simpson has repeated these results in Calcutta, and a similar and more absolutely unequivocal result has been attained by Klein, who in 1892 took smallpox lymph and inoculated a series of calves, producing ultimately an atypical vaccinia. But with lymph from this source Cory inoculated a large number of children, producing in every case typical vaccinia, and the retro-vaccination of this humanised lymph on calves gave absolutely typical results. By the kindness of Dr. Klein I am able to pass round photographs of some of these children, so that you may judge for yourselves as to the characters of the local eruption produced. To my mind such results prove to absolute demonstration the identity of vaccinia as modified variola. These later experiments have been performed in the full light of modern pathological knowledge, by the most highly qualified observers, and with the strictest precautions, and rest, therefore, on a more unassailable basis than the results attained in the early part of the century.

* This paper makes no pretence at originality. It is merely an attempt to set forth some of the facts and arguments on the subject for the benefit of students and others who have not the time to study its literature for themselves. I have to acknowledge the assistance derived from McVail's writings, and from the Report of the Vaccination Commission; the facts about the Gloucester epidemic are taken from Dr. Bond's pamphlet, published by the Jenner Society. The crushing reply to the minority report of the Dissentient Commissioners recently delivered by Dr. McVail at the Epidemiological Society was subsequent in date to the reading of this paper; my own comments are feeble in comparison.—F. W. A.

(4) I may add, though with more diffidence, a further argument. Until recently no one had succeeded in demonstrating the nature of the actual virus of variola or vaccinia. Such demonstration is still incomplete, but within the past few years Klein, and subsequently Copeman, have shown that it is possible to demonstrate in very early smallpox lymph a minute spore-bearing bacillus, which they regard as the probable cause of the disease. Should this bacillus be successfully cultivated, and should it then prove capable of producing vaccinia when inoculated in calves, the demonstration will be complete. Meanwhile the importance of the fact for our present argument rests in the consideration that the bacillus is demonstrable equally in variolous lymph and that of vaccinia, and as it differs entirely from any other known bacillus some degree of weight may be attached to its presence.

Considering the question, therefore, in the light of modern pathology, I think we are justified in affirming that cow-pox or vaccinia is neither more nor less than a modified form of variola, comparable with what we know of the attenuations of other specific poisons. It has been complained that no definition of vaccination has been attempted. I venture to define it as "implantation of the virus of smallpox, attenuated by bovine transmission, with the view of producing a localised affection which can nevertheless confer an immunity of some considerable duration against the acute disease."

II. I now pass to the second question—what degree of immunity does vaccinia confer against variola? The answer may be sought in two groups of facts.

(1) In the early days of vaccination, when inoculation with smallpox was an everyday thing, the question was answered by actual experiment on the person, and reliance was very properly placed on this "variolous test." Inoculation of smallpox was known to succeed in the vast majority of persons unprotected by previous smallpox. The variolous test was found to yield no result in the vast majority of vaccinated persons. The conclusion that vaccinia protected against variola was plain, and from what was known as to the permanence of the protection given by a previous attack of smallpox, it was naturally enough assumed at first that the protection given by vaccinia would be equally lasting. Only the lapse of a sufficient number of years could prove the fallacy of this assumption; but it soon became recognised that smallpox could and did occur in vaccinated persons, though usually only after many years: it was further noted that post-vaccinal smallpox was usually mild in type. At the present day, of course, it is universally recognised that the full protective influence of vaccination lasts only some seven to ten years, and thenceforwards gradually fades, though its influence in mitigating the disease may be traced for a considerable period of life. Further, it is well known that individuals vary widely in the duration of their protection, presumably in accordance with their degree of natural susceptibility to variola.

(2) The variolous test is now illegal, and we have to rely on statistical evidence instead of on experiment. We have a century of experience on which to found our judgment. Our task is to eliminate all possible fallacies, and correctly to interpret the vast mass of statistics which has accumulated. The evidence falls under two main heads: (*a*) the diminished incidence of and mortality from smallpox generally since the introduction of vaccination; and (*b*) comparison of the smallpox incidence and mortality upon the vaccinated and unvaccinated respectively in given local epidemics.

In the early years of vaccination smallpox was much commoner than it is now, and no trouble was taken to isolate cases. The practice of inoculation, too, may sometimes have served to spread infection. People were hence very much more exposed to infection from smallpox than they are now, and this made the difference between vaccinated and unvaccinated persons as regards their susceptibility, a striking and apparent fact of everyday life. Nowadays the comparative rarity of smallpox, and the strict isolation practised, combine to render exposure to its infection a relatively uncommon thing. Hence the contrast between vaccinated and unvaccinated has ceased to be a conspicuous and public phenomenon, and the very success of vaccination in diminishing smallpox has come to be a cause of its neglect among the uneducated and misinformed. It is only when an extensive outbreak occurs, and exposure to infection becomes general, that the lesson is taught anew in its full force.

Before discussing the data furnished by recent epidemics let me draw your attention to the diagram on the wall, which has been enlarged from that given by McVail in the treatise on Public Health edited by Stevenson and Murphy. It shows the smallpox mortality in London from the year 1630 onwards. From 1838 the figures are taken from the Reports of the Registrar-General; before that they are calculated from the London Bills of Mortality. The diagram represents not the actual total of smallpox deaths in each year, but the number of smallpox deaths per 1000 total deaths from all causes. The increase of population is thus avoided as a disturbing factor. The old Bills of Mortality were probably inadequate enough when judged by modern standards, and there is reason to suppose that they understated the total smallpox mortality, but they form by far the most important evidence we possess as to smallpox mortality in the seventeenth and eighteenth centuries, and the information they afford as to the proportion of smallpox deaths to total deaths is probably much more accurate and reliable than their statements as to total smallpox mortality.

One of the most striking features seen in the diagram is the fluctuation of the number of deaths from year to year. It can be seen that each year of excessive mortality is commonly followed by a year of much diminished mortality, and that three or four years more elapse before another year of very high mortality is reached. There is, in fact, a rough sort of periodicity about smallpox, a high degree of epidemic prevalence recurring every four or five years. The explanation of this fact is not very difficult. Before Jenner's time smallpox was, at least in towns like London, where it was constantly present, mainly a children's disease. Even up to 1853 more than 80 per cent. of smallpox deaths in London were in children under ten, and in Geneva in the seventeenth and eighteenth centuries over 96 per cent. The effect of a big epidemic was roughly to kill or immunise this eminently susceptible section of the population, and it was not till a fresh soil had been prepared for the disease by the births of a few years that another big epidemic was possible.

A second striking feature in the diagram is the fact that smallpox was much more prevalent in the eighteenth than in the seventeenth century, or at least that it killed many more people. An explanation which has been held to account for this is the introduction and spread of the practice of inoculation, which was first performed in England in 1721, but did not prevail much till after 1740. During the latter half of the century the celebrated inoculator, Sutton, claimed to have inoculated 100,000 people. Though the attack induced was habitually a mild one, it nevertheless served as a centre of infection to the unprotected. Where there were many unprotected the result may have been to increase smallpox. Where there were few, as must have been the case in London, it is likely that the practice did more good than harm. On the whole there seems no sufficient evidence to attribute the increase in smallpox in London in the eighteenth century to the practice of inoculation alone, and I know of no other reasonable explanation which has been advanced.

Let us turn now to the astonishing diminution in smallpox mortality which commenced with the closing year of the last century. Vaccination was introduced in 1798. During the first quarter of the present century the diminution of smallpox mortality is seen to be marked, and on the whole progressive. During the remainder of the century the decrease is still more apparent; only twice, in 1838 and 1872, has the disease approached in its mortality that which was the rule in the eighteenth century. The obvious inference is that it was the gradual spread of the practice of vaccination which was accountable for the improvement. Unless other adequate ground can be shown for it this explanation must be inevitably accepted. The only other tenable ground which can be alleged is that which has been adopted in the minority report of the dissentient members of the Vaccination Commission. It is as follows:—If we examine the statistics of the deaths from "fever" (*i.e.* typhoid, typhus, and simple continued fever), it will be found that they have declined very nearly in the same proportion as the deaths from smallpox. Typhus fever has declined even more than smallpox. No one pretends that any influence has been at work in the case of fever other than simple sanitation. The inference is that improved sanitation has been responsible also for the decline of smallpox. The prevalence of smallpox was kept up abnormally during the eighteenth century by the practice of inoculation; the substitution of vaccination, which was non-contagious, for inoculation, which was contagious, must inevitably have produced a great effect on the smallpox death-rate, apart from any immunity it conferred. This is the argument; let us now examine it.

If we consider the death-rates from the different specific fevers during the present century, which period practically covers the rise and progress of modern sanitation, we notice very astonishing differences between them. Some have shown a remarkable decrease: typhus has been practically stamped out in London; typhoid mortality has fallen by more than one half in the last thirty years; the decline of scarlet fever is equally even more striking. In contrast to these, some, such as measles, whooping-cough, and diphtheria, have remained either unchanged, or have even increased; the progress of sanitation has done nothing to check them. It is very easy to select typhus and typhoid, and to compare smallpox with them, and show that they have declined equally, and hence to infer that the same

influence has been at work in the production of the decline in each case. But it is equally easy to select measles or whooping-cough as the standard of comparison, and to arrive at an absolutely reverse conclusion. We have at least as much right to select the latter as the former, and indeed I think more right, for typhus and typhoid are essentially filth diseases, and are much less comparable with smallpox than are scarlet fever or measles. The argument hence loses its force to a very considerable extent, and there are other considerations which weaken it still further. It will be seen that the main decline in London smallpox occurred during the first quarter of the century. A survey of the history of sanitation, such as that presented in Sir John Simon's admirable book, shows nothing more clearly than this, that sanitation has grown up almost exclusively in the reign of Victoria. It is true that during the eighteenth century people were beginning to realise the preventability of much of the prevalent zymotic and other disease, but nothing was done. It was not till the reign of William IV that Local and Central Boards of Health were instituted, and these merely inspected and advised; when Queen Victoria ascended the throne there was practically no sanitary legislation at all. The real beginnings of modern sanitation were the Inquiry, lasting from 1839 to 1842, with which Sir Edwin Chadwick's name is so prominently associated, the Royal Commission which resulted, and ultimately the Public Health Act of 1848. The great mass of sanitary progress was therefore subsequent to the decline in smallpox, and it will be seen to have produced a very trivial effect upon smallpox mortality compared with the earlier decline. It may be answered that, notwithstanding this, "fever" had actually declined during the eighteenth century to an enormous extent. According to the Bills of Mortality it stood answerable for an average mortality of 785 per 100,000 living before the middle of the eighteenth century, and had dropped to 264 in the first decennium of the nineteenth. But before allowing this comparison we must remember that the causes of death were returned by ignorant old women known as "searchers;" now smallpox was a well-known and distinctive disease, easily recognisable even by the ignorant, while "fever" was a *caput mortuum* of miscellaneous zymotic and septic diseases which may have included almost anything. The progress of medical knowledge has weeded it down nowadays to typhus, typhoid, and simple continued fever, but Heaven only knows what these old women meant by it in the last century. The number of deaths returned as due to it would naturally diminish with the growth of knowledge, and hence the apparent decline in its mortality must be very largely discounted when contrasted with that of a definite disease like smallpox. The argument, in fact, cannot be taken seriously. We have no satisfactory proof that sanitation can have exercised upon smallpox the extraordinary influence necessary to explain the fall which took place in the first quarter of the present century. If further argument be needed, let us turn to the facts from abroad. Vaccination was introduced into many other European countries within a year or two of its inception in England. From some of these countries, *e. g.* Sweden and Denmark, we have full statistics of smallpox mortality for this and part of the last century. Everywhere the same result is apparent—an immediate and startling decrease in smallpox mortality. Now England enjoys the credit of being the pioneer in sanitation amongst the countries of Europe: other nations followed us, and usually at a considerable interval. If, then, it were sanitation which explains the diminution in smallpox which marked the first quarter of this century, the diminution should have been later in other countries than in England. It was not. In every case it followed the introduction of vaccination as an immediate and apparently direct result.

There is another and still more powerful argument which may be used against those who attribute the decline in smallpox to sanitation alone. The Registrar-General has calculated out the mean annual deaths from "fever" of successive periods of life per million living at each age-period, and has done this for three different terms of years, from 1847 to 1853, thence to 1871, and thence to 1887. The resulting figures show with marvellous uniformity a steady decrease which is practically the same for each period of life, the decline for the first five years of life being only very slightly more marked than that for all ages taken together. This is what we expect sanitation to do, viz. to produce its beneficial effects upon people of all ages, with a special reduction of infant mortality (which appears less in the case of "fever" than it would elsewhere, because this is not a group of diseases specially fatal in children, but rather the reverse). If now we contrast a similar table furnished by the Registrar-General with regard to smallpox for the same periods, a very different state of affairs is disclosed. We find that, under the age of five years, smallpox deaths have fallen from 1617 per million to 242; between five and ten years of age from 337 to 120. From ten to fifteen years the decline is trivial, from 94 to 69; while from fifteen years upwards there has been a positive rise in the annual number of smallpox deaths (per million living at ages above fifteen). Now these terms of years were chosen because up to 1853 vaccination was optional only, from thence to 1871 it was obligatory but insufficiently enforced, and thence to 1887 much more efficiently enforced. It will be seen, then, that the total reduction in smallpox mortality which has occurred under the vaccination laws has been the resultant of an enormous decrease in child mortality, with a positive increase in adult mortality. We are asked to believe that sanitation has done this, and invited to compare the decline in "fever" with that in smallpox; nor can it be denied that the comparison is instructive when pursued in detail.

No sane man will deny that improved sanitation, by diminishing overcrowding and improving ventilation, must have done much to diminish the opportunities for the spread of smallpox, and by raising the general average of health may have done something to increase resistance against it. But that it has been the main agent in its decline appears to me disproved by the considerations I have mentioned, viz. that the decline was immediately visible on the introduction of vaccination, and was long anterior in date to the main sanitary advances of the century; that a similar decline occurred in other countries, apart from any sanitary improvements, as soon as vaccination was introduced, and that the nature of the decline was altogether peculiar, being confined to the early years of life, while the adult mortality has actually risen—a state of affairs easily explicable when we remember the temporary character of the protection afforded by vaccination, but absolutely at variance with what we know of the mode in which sanitation affects the death-rate.

Let us turn now to the other part of the argument, that the decrease which followed vaccination was due to the concomitant decrease in the practice of inoculation. The supposition that inoculation was in the main accountable for the increase in the prevalence of smallpox which marked the eighteenth century is at best an uncertain one.

On *a priori* grounds it seems likely, as I have said, that inoculation must have served to spread the disease amongst an ill-protected population. But the population of London and other large towns at the close of the last century was a well-protected one: probably some 80 per cent. were protected either by acquired or inoculated smallpox. I cannot therefore believe that in London inoculation could have been so largely accountable for the high smallpox mortality in the eighteenth century as this hypothesis demands. In some towns, as in Boston, U.S.A., it is known that the introduction of inoculation very largely diminished smallpox mortality. Another argument of importance is this: if inoculation were responsible for the increase in smallpox mortality in the eighteenth century, it should be demonstrable that this increase went hand in hand with the spread of that practice. It should not have been marked till 1740 and the succeeding years, and should have risen considerably during the latter half of the century as inoculation became commoner. A glance at the diagram will show that this is not borne out by facts. While, therefore, not disputing that some part of the decline in smallpox during the first quarter of the nineteenth century may have depended on the decrease in inoculation, I consider that it is not demonstrated that any important share in the decline is due to that cause. There remains the view that it was due to the introduction and spread of vaccination. I have dealt with the arguments advanced by the dissentient commissioners at some length because they are well worth argument, as being the only serious attempt to explain away the decrease of smallpox on the introduction of vaccination.

Let us now pass on to the other and even more striking statistical evidence derived from a comparison of the vaccinated and unvaccinated in given epidemics. I would first invite your attention to the natural fatality of smallpox, *i.e.* the case-mortality apart from inoculation or vaccination. We know that when introduced into new countries its fatality is enormous, exceeding 50 per cent. of those attacked in some cases. But setting this aside, we know that the records of the London Smallpox Hospital showed in the last century a fatality of 25 per cent., and during the last quarter of the century 32 per cent. This is probably too high for the average fatality, as only the more severe cases would have been treated in hospital. It is clear, too, that the fatality varied considerably in different epidemics, and we have no sufficient data for estimating the average fatality of the disease. One point, however, comes out very clearly—the enormous share in the smallpox death-rate borne by very young children. In the epidemic at Chester in 1774 all the 202 deaths were in children under ten, and in an epidemic at Warrington in 1773 they were all in children under nine. In every epidemic in which

data exist showing the ages at death the mortality is seen to be mainly amongst infants. McVail states that, summing up the statistics of smallpox deaths from Geneva, the Hague, Kilmarnock, Edinburgh, Manchester, Warrington, and Chester for former centuries, we find that, out of 36,755 deaths, no less than 17,252 were under two years of age. These facts prove that in times anterior to the introduction of vaccination fatal smallpox was essentially a phenomenon of childhood, and especially of the first two years of life.

Within the past ten years several more or less extensive outbreaks of smallpox have occurred in England, and have been the subject of critical investigation. Even here, with the greatest precautions, statistics are in a measure fallible, though to a limited degree only. A vaccination census must depend upon either the statements of individuals, sometimes inaccurate, or upon the presence or absence of scars, sometimes obliterated or obscured by eruption. In epidemic times a condition of panic arises in which vaccination is largely resorted to while the epidemic is at its height, and this may tend to confuse the issue. The statistics, however, are so overwhelming that they can stand very extensive reductions for possible errors under these heads without any material weakening of the case for vaccination. On the other hand, allowance must be made in the opposite direction. Since the protection given by vaccination is not permanent, those adults who have been vaccinated in infancy only are imperfectly protected. These form a very considerable section of the population, and hence it commonly happens that in a given epidemic an actually larger number of vaccinated than unvaccinated are attacked. This is put forward by the opponents of vaccination as an argument against the practice, whereas in fact it can be shown easily enough to be an argument in favour of re-vaccination. Statistics require proper analysis before just conclusions can be drawn from them, and smallpox death-rates require to be studied in relation to age-periods.

I will select the figures of the late outbreak at Gloucester as a typical instance of this, though in fact the statistics of other outbreaks would serve as well. That at Gloucester, however, is the most recent epidemic of any importance, and is peculiarly instructive as showing the effects which may be produced in a relatively unvaccinated town.

Gloucester is a city of some 42,000 inhabitants, not behind other similar towns in its general sanitary condition. For some years previous to the epidemic a local anti-vaccination society had established itself there, and had attained such influence that in 1887 the Board of Guardians elected to discontinue vaccination prosecutions. The result was a practical suspension of vaccination in the city. The total vaccinations, which in 1886 had been 1095, fell till in 1891 they numbered only thirty-four. In the ten years from 1886 to 1895, only 2378 vaccinations were performed, out of 15,682 children born; 3176 had died, so that at the end of 1895 there was a balance of over 10,000 unvaccinated children. The adult population was fairly well vaccinated, for prior to the propaganda of the anti-vaccination society the law had been observed in Gloucester as well as in most other parts of England. The isolation resources of the City Council were not very extensive. Under these conditions smallpox was imported into the city towards the end of 1895. A case in a child was concealed and treated as measles, and in a short time the disease had grown out of all control; isolation became impossible, and in April, 1896, when the outbreak was at its worst, over 200 cases were occurring per week. The city was practically boycotted. It was only by prodigious efforts that the machinery of vaccination was at length brought efficiently into play. Mainly by persuasion, 36,000 persons were vaccinated or re-vaccinated. The effect on the epidemic was immediate and striking. By the end of July smallpox was stamped out, and probably no epidemic of equal magnitude has ever been so promptly suppressed. The conversion of the anti-vaccinationists was astounding; few remained, and there is now no city in England so well vaccinated as Gloucester. The cost to the city was estimated by the President of the Gloucester Chamber of Commerce at over £150,000. Such is the broad general lesson.

The statistics of the epidemic, to which I would specially draw your attention, are shortly as follows:—The total number of cases in the city and suburbs was 2035, *i. e.* about 5 per cent. of the population suffered in the course of a few months—a degree of severity almost unexampled amongst recent epidemics. Of these cases, 1208 occurred in those vaccinated in infancy, and 789 in unvaccinated persons; in thirty-eight cases the question of vaccination was uncertain.

At first sight these figures might be claimed as unfavorable to vaccination, but an examination of the age incidence of the attacks soon sets the matter in a very different light. Of the 1208 attacks in persons vaccinated in infancy, 916, or over 75 per cent., occurred in persons over twenty, and only 23, or less than 2 per cent, in persons under ten years of age. Of the 789 attacks in unvaccinated persons, 699, or over 88 per cent., occurred in children under ten, and only 39, or less than 5 per cent., in persons over twenty. The ages ten to twenty years occupy an intermediate position. The inference as to the protective effect of vaccination, for a period of at least ten years, is irresistible, even if we allow a large reduction on the grounds which I mentioned previously.

Still more obvious are the fatality statistics. Out of a total of 443 deaths, 113 were contributed by those vaccinated in infancy, and 313 by the unvaccinated, in spite of the smaller number of attacks in the latter class. Not a single child under ten died amongst the vaccinated, and 281 children under ten died amongst the unvaccinated: 40 per cent. of the unvaccinated children who were attacked died.

A more minute analysis of the figures only confirms the conclusion that the protection afforded by infant vaccination is enormous for the first ten years of life, and thence gradually fails, but that it endures in some degree certainly for fifty years.

The statistics afforded by the unvaccinated population of Gloucester show neither more nor less than a return to the conditions prevailing in the eighteenth century, and prove as clearly as possible that vaccination, and not sanitation, has been concerned in the reduction of smallpox mortality which has prevailed since Jenner's day.

I would not weary you with figures. Those of other recent epidemics, amongst which I would specially instance those worked out by Barry in regard to the Sheffield outbreak, all tell a precisely similar tale. Where the proportion of unvaccinated children in the community attacked is high, there the infant mortality is high; where it is low, the child mortality is less predominant. I cannot conceive that any logical mind can resist the inference.

Accurate evidence on the effect of re-vaccination is less easy to obtain because of the absence of statistics; but where it is uniformly enforced its effects are striking. In Prussia re-vaccination was made compulsory in 1874, with the result that smallpox at once almost disappeared from the country. In our own army and navy compulsory re-vaccination has immensely reduced the smallpox mortality. Perhaps more striking than any other figures are those relating to nurses and attendants at smallpox hospitals. A committee of the Epidemiological Society reported on 734 nurses and attendants at the Metropolitan Asylums Board Hospitals: seventy-nine had suffered from previous smallpox, and none of these were attacked; 645 had been re-vaccinated, and none of these were attacked; ten had not been re-vaccinated, and every one caught the disease.

Had I time I could go on to give you figures showing the much smaller fatality of smallpox in cases occurring amongst the vaccinated than amongst the unvaccinated; and to cite the figures of Marson and Gayton, showing that the fatality amongst the vaccinated varies inversely with the number, extent, and character of the cicatrices. But I fear my time and your patience are alike exhausted. I must be content if I have been able to present you with some of the main facts upon which a rational belief in the efficacy of vaccination may be founded, and an examination of some of the more important arguments which have been adduced against it. Put briefly, the effects of efficient vaccination seem to be an almost absolute protection for the first ten years of life, a protection which has succeeded in absolutely reversing the natural age incidence of fatal smallpox, and, by sweeping away the former enormous child mortality from the disease, has reduced its total mortality to a mere fraction of its former extent. On the other hand, it has somewhat increased adult smallpox mortality, since the protection afforded to adults by vaccination in infancy is less than that which was formerly afforded by the usual attack of smallpox in childhood. There is reason for believing that re-vaccination would not only prevent this increase, but practically stamp out the disease.

Let me, in concluding, touch very briefly on the objections which are raised against the practice on the ground of its risks. Vaccination is undoubtedly attended by certain dangers, like any other scratch on the skin. That pure vaccinia should cause death must be an event of the most extreme rarity if it ever occurs; the vast majority of deaths returned as due to vaccination are deaths from its complications, *i. e.* dependent on concomitant or subsequent infection with other micro-organisms It is practically impossible to obtain vaccine lymph free from pyogenic cocci, or under ordinary conditions to prevent their access to the vesicles. The severity of local inflammation thus induced is sometimes accountable for death in weakly infants. Erysipelas, again, may occur, and here dirt and carelessness are probably often to blame, though not always. The same is true

of other septic complications. It is said that impetigo contagiosa may sometimes be transmitted in arm-to-arm vaccination; the experience of children's hospitals shows that troublesome and intractable forms of eczema and other cutaneous diseases occasionally, though not often, date from vaccination. But the great risk of which so much capital is made by opponents of the practice is vaccino-syphilis. It is admitted that syphilis is occasionally, though very, very rarely, thus transmitted. The great majority of reputed cases are probably inherited syphilis, which commonly manifests itself about the second or third month at or soon after the time when vaccination is habitually performed. The true test is, of course, whether or not a genuine primary chancre appears, after the usual incubation period, at the seat of the vaccinal inoculation, and is followed later by secondary symptoms; and this has sometimes been the case. It is this risk, infinitesimal though it be, which has induced the Vaccination Commissioners to recommend that calf-lymph should be used whenever the parents desire it. But let us see what all these risks amount to. Some fifty deaths, on an average, are ascribed per annum in England and Wales to vaccination,—not all of these, perhaps, with justice. The sum total of annual primary vaccinations in England and Wales is between 700,000 and 800,000. Against this insignificant number of deaths we have to set the enormous infant mortality from smallpox in the last century, exact figures for which cannot be given, but of which you may form a rough estimate from the diagram on the wall, by remembering that between 80 and 90 per cent. of the mortality there indicated in London alone was in children under ten. You must not, of course, expect parents who have lost a child as the result of vaccination to take this dispassionate view of the facts, but it is your duty and that of Government to weigh the gain against the loss, and be guided accordingly.

Granting, however, that you and the vast majority of intelligent persons are convinced by the facts which I have mentioned, and the far more numerous arguments which I have had no time to discuss to-night, that vaccination is an indispensable prophylactic against smallpox, there remains the purely ethical question, what right has the majority to enforce its convictions upon the minority who do not agree with them? I do not propose to discuss this matter to-night. I will only say, in closing, that since the matter does not merely concern the health of the individual, but that of the community at large, the majority has, in my opinion, an absolute right to enforce its views, for its own protection, as it does in all other social relations. As to the expediency of so enforcing them, opinions may well differ. For my own part I am disposed to place more reliance upon an active policy of instruction and education amongst the ignorant, who have hitherto been at the mercy of the irresponsible anti-vaccinationist, than upon a system of pains and penalties, which has of late succeeded only in evoking amongst a certain class an unreasoning hostility, begotten by prejudice out of ignorance, and baptised by pseudo-martyrdom.

Notes.

The Kirkes Scholarship and Gold Medal have been awarded to Hugh Thursfield, *Proxime accessit*—T. J. Horder.

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The first Festival Dinner of the Alexandra Hospital for Children with Hip Disease was held on Thursday, April 8th, at the Whitehall Rooms, Hôtel Métropole, the Duke of Fife in the chair. The occasion was in every way a success; most distinctly so from a financial point of view, close upon £3000 being promised towards the funds of the hospital. The toast of the Medical, Surgical, and Nursing Staff was replied to by Mr. Bowlby in an excellent and entertaining speech. Though our profession has nothing to do with politics, yet, said he, we combine the merits of all the three great political parties; for we are liberal in our medicine, conservative in our surgery, and radical in our cures.

We have been requested to call attention again to the claims of Ernest Guy Robertson, aged eleven years, for the votes of Governors of the Royal Medical College, Epsom, at the ensuing May election. He is the son of an old Bart.'s man, who was in practice at Bradford, and subsequently at Hambledon, Hants. His father died in 1891, leaving a widow and four children with very small means. The case is strongly recommended by Dr. Symes Thompson, Dr. Sansom, and other well-known medical men. Proxies should be sent to Mrs. Robertson, St. David's, Kingston Crescent, Portsmouth.

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We cull the following precious paragraph from the advertisement columns of a morning paper:

"The Society for the Protection of Hospital Patients appeals for Funds to carry on its work, namely, the Protection of Hospital Patients from unnecessary operations and experiments. For particulars apply to the Secretary, A. J. Beatty, 40, Leadenhall House, E.C."

A lady promptly applied by letter to the Secretary of this mysterious Society for particulars, and, strange to say, none were forthcoming. Comment is unnecessary.

* * *

Mr. W. J. Walsham has been elected Joint Lecturer on Surgery *vice* Mr. H. T. Butlin resigned.

* * *

The Honorary Medal of the Royal College of Surgeons has been awarded to Sir James Paget.

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Mr. R. C. Bailey has been awarded the Jacksonian Prize of the Royal College of Surgeons for his dissertation bearing the motto "There's the respect that makes calamity of so long life"—*Hamlet*, Act III, Sc. 2. The subject of the essay was "The pathology, diagnosis, and treatment of diseases of the prostate gland."

* * *

For three years in succession the Jacksonian prize has been gained by a teacher in our Medical School; Mr. Waring in 1895, Dr. Kanthack last year, and Mr. Bailey this year.

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Sir Dyce Duckworth and Dr. Lauder Brunton have been nominated as delegates for the Royal College of Physicians at the International Medical Congress to be held at Moscow.

Amalgamated Clubs.

NEW MEMBERS.

H. V. Wenham.	J. H. Harker.
F. Gröne.	F. W. Jackson.
F. H. M. Spaight.	R. J. P. Thomas.
C. P. Charles.	W. R. L. Drawbridge.
W. P. S. Branson.	G. F. Gill.

CRICKET CLUB, 1897.

The coming season promises to be one of the best, if not *the* best we have had for many years. All our last year's men will be

available, and many of them will be able to play more regularly than in the past season. Mr. J. A. Willett's wrist has also quite recovered from the unfortunate accident which put him on the sick list for the last half of the season. The ground at Winchmore Hill looks as if it would turn out very well, and as we have had plenty of rain to keep it going, all we have to do is to pray for fine weather. There has been rather an unfortunate misunderstanding on account of the United Hospitals Club not being able to inform us (as their meeting is not held till May) whether our last year's captain, Mr. H. Bond, is still in his year. This has, of course, prevented us having our match list printed, and also prevented us from selecting our captain for this year; this will no doubt, however, be cleared up, and we may safely look forward to a really good season. Last year it will be remembered we drew with Thomas's for the Cup, and that match is to be played off early in May. The fixtures were published in the last JOURNAL.

Junior Staff Appointments.

HOUSE PHYSICIAN TO—

	SENIOR.	JUNIOR.
Dr. Church	G. Wedd, M.A., M.B., B.C.(Cantab.).	A. L. Ormerod, M.A., M.B., B.Ch.(Oxon.), M.R.C.S., L.R.C.P.
Dr. Gee	G. A. Auden, B.A., M.B., B.C.(Cantab.), M.R.C.S., L.R.C.P.	T. J. Horder, B.Sc. (Lond.), M.R.C.S., L.R.C.P.
Sir D. Duckworth	L. B. Burnett. M.A., M.B., B.C.(Cantab.), M.R.C.S., L.R.C.P.	J. Hussey, M.B.(Lond.), M.R.C.S., L.R.C.P.
Dr. Hensley	T. Hampton, M.R.C.S., L.R.C.P.	F. H. Maturin, B.A., M.B., B.C.(Cantab.), M.R.C.S, L.R.C.P.
Dr. Brunton	W. F. Cross, M.R.C.S., L.R.C.P.	E. G. D. Drury, M.B., B.S.(Lond.), M.R.C.S., L.R.C.P.

HOUSE SURGEON TO—

Mr. Smith	J. E. G. Calverley, M.R.C.S., L.R.C.P.	H. W. Lance, B.A., M.B., B.C.(Cantab.).
Mr. Willett	J. W. Haines, M.B., B.S.(Lond.), F.R.C.S., L.R.C.P.	J. A. O. Briggs, M.B. (Lond.), M.R.C.S., L.R.C.P.
Mr. Langton	M. G. Pearson, M.B., B.Sc.(Lond.), M.R.C.S., L.R.C.P.	E. Laming-Evans, M.A., M.B., B.C.(Cantab.), M.R.C.S., L.R.C.P.
Mr. Marsh	H. B. Meakin, M.D. (Lond.), M.R.C.S., L.R.C.P.	J. J. Grace, M.B, B.S. (Durham), F.R.C.S., L.R.C.P.
Mr. Butlin	J. P. Maxwell, M.R.C.S., L.R.C.P.	H. Williamson, B.A. (Cantab.), M.R.C.S., L.R.C.P.

OPHTHALMIC HOUSE SURGEON.—
E. W. Brewerton, M.R.C.S., L.R.C.P.

RESIDENT MIDWIFERY ASSISTANT.—
E. W. Ormerod, M.A., M.B., B.C.(Cantab.), M.R.C.S., L.R.C.P.

EXTERN MIDWIFERY ASSISTANT.—
April—W. Langdon Brown, M.A., M.B., B.C.(Cantab.).

RESIDENT ASSISTANT ANÆSTHETISTS.—
Senior.—H. J. Paterson, M.A., M.B., B.C.(Cantab.), M.R.C.S., L.R.C.P.
Junior.—F. H. Lewis, B.A., M.B., B.C.(Cantab.), M.R.C.S., L.R.C.P.

Appointments.

COWIN, D. H. F., M.R.C.S., L.R.C.P., appointed Assistant House Surgeon to the Devon and East Cornwall Hospital.

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TICKELL, H. M., M.A., M.B., B.C.(Cantab.), appointed House Physician to the Brompton Hospital for Diseases of the Chest.

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TUCKER, A. B., M.B.(Lond)., M.R.C.S., L.R.C.P., appointed House Surgeon to the Children's Hospital, Shadwell.

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WRANGHAM, W, M B.(Lond.), M.R.C.S., L.R.C.P., appointed Assistant House Surgeon to the Leicester Infirmary.

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HUGGINS, S. P., M.B (Lond.), M.R.C.S., L.R.C.P., appointed House Physician to the Sussex County Hospital, Brighton.

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DRAGE, LOVELL, M.A., M.D.(Oxon.), M.R.C.S., appointed Coroner for the St. Albans division of Hertfordshire.

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DUCAT, ARTHUR D., M.B.(Lond.), M.R.C.S., L.R.C.P., appointed District Medical Officer and Public Vaccinator to the Chiswick district of the Brentford Union.

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HOFMEISTER, H. EDGAR, B.A., M.B., B.C.(Cantab.), M.R.C.S.(Eng.), appointed Medical Officer of Health to the East Cowes District Council.

* * *

LOW, CHARLES W., M.B., D.P.H.. F.C.S., appointed Medical Officer of Health to the Stowmarket Urban District Council.

* * *

COLLINGRIDGE, W., M.D., D.P.H., M.R.C.S., reappointed Medical Officer of Health for the Port of London.

* * *

NUNN, P. W. G., L.R.C.P., M.R.C.S., reappointed Medical Officer of Health to the town of Bournemouth.

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KINGDON, J. R., B.A.(Camb.), M.R.C.S., L.R.C.P., House Surgeon to the Weston-super-Mare Hospital and Dispensary.

Cases of Special Interest.

Mark, bed 11.—Acute rheumatism and morbus cordis.
Mark, bed 1.—Functional paraplegia.
Luke, bed 8.—Paroxysmal tachycardia.
Matthew, bed 7.—Aneurism of innominate artery.
Matthew, bed 11.—Right hemiplegia.
Colston, bed 2.—Left hemiplegia.
Colston, bed 18.—Purpura.
Colston, bed 20.—Simple meningitis.
Rahere, bed 17.—Sarcoma of chest wall.
Rahere, bed 22.—Postero-lateral sclerosis.
Rahere, bed 24.—Alcoholic neuritis.
Faith, bed 2.—Exophthalmic goitre.
Faith, bed 9.—Exophthalmic goitre.
Faith, bed 11.—Locomotor ataxy.
Faith, bed 20.—Basal meningitis.
Hope, bed 4.—Right hemiplegia (following scarlatina).
John, bed 9.—Hydrochloric acid poisoning.
Mary, bed 2.—Chorea.
Mary, bed 10.—Chorea.
Mary, bed 14.—Hydronephrosis and morbus cordis.
Elizabeth, bed 24.—Rheumatoid arthritis (æt. 5).

The Month's Calendar.

MAY.

Sat. 1st.—Summer Session begins. Names for Brackenbury Medical and Surgical Scholarships to be sent in. Cricket Nets and Tennis Courts open for play.
Tues. 4th.—Dr. Hensley's and Mr. Marsh's duty. Dr. Thorne-Thorne commences his course of Public Health Lectures, 10.30, Med. Th.
Wed. 5th.—Dr. Claye Shaw commences his Lectures on Mental Diseases, 11.30, Med. Th. Sale of Papers, Abernethian Society, 12.30 p.m. Clinical Lecture, Mr. Smith.
Thurs. 6th.—Primary Fellowship Exam. Brackenbury Medical Sch. Exam.
Fri. 7th.—Brackenbury Surgical Sch. Exam. Dr. Lauder Brunton's and Mr. Butlin's duty. Clinical Lecture, Dr. Church.
Tues. 11th.—Dr. Church's and Mr. Smith's duty.
Wed. 12th.—View-day.
Fri. 14th.—Dr. Gee's and Mr. Willett's duty. Clinical Lecture, Dr. Gee.
Tues. 18th.—Sir Dyce Duckworth's and Mr. Langton's duty.
Wed. 19th.—Clinical Lecture, Mr. Smith.
Thurs. 20th—Lawrence Sch. Exam.
Fri. 21st.—Dr. Hensley's and Mr. Marsh's duty. Clinical Lecture, Sir Dyce Duckworth.
Tues. 25th.—Dr. Lauder Brunton's and Mr. Butlin's duty.
Wed. 26th.—Clinical Lecture, Mr. Langton.
Fri. 28th.—Dr. Church's and Mr. Smith's duty. Clinical Lecture, Dr. Hensley.

Clinical Lectures for the Summer Session.

Clinical Medicine.—Fridays at 1 p.m.

May 7th.—Dr. Church.
„ 14th.—Dr. Gee.
„ 21st.—Sir Dyce Duckworth.
„ 28th.—Dr. Hensley.
June 4th.—Dr. Brunton.
„ 11th.—Dr. Church.
„ 18th.—Dr. Gee.
„ 25th.—Sir D. Duckworth.
July 2nd.—Dr. Hensley.
„ 9th.—Dr. Brunton.

Clinical Surgery.—Wednesdays at 2.45 p.m.

May 12th.—Mr. Smith.
„ 19th.—Mr. Smith.
„ 26th.—Mr. Langton.
June 2nd.—Mr. Langton.
„ 9th.—Mr. Marsh.
„ 16th.—Mr. Marsh.
„ 23rd.—Mr. Marsh.
„ 30th.—Mr. Butlin.
July 7th.—Mr. Butlin.
„ 14th.—Mr. Butlin.

Examinations.

First Conjoint.—*Four years regulations. Chemistry.*—F. B. Jones. *Materia Medica.*—E. F. Crabtree.

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First Conjoint.—*Five years regulations. Chemistry and Physics.*—A. Butler, V. J. Duigan, W. J. G. Johnson, C. H. Turner, F. J. Wood.

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First Conjoint.—*Pharmacy.*—G. G. Campbell, C. C. Morgan, C. C. B. Thompson.

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First Conjoint.—*Biology.*—H. H. Butcher, F. W. Cheese, E. W. Dall, G. S. Ewen, L. C. Ferguson, P. Harris, E. O. Hughes, H. Mills, L. M. Morris, A. F. C. Pollard, E. W. Price, H. E. Scoones, M. B. Scott, H. Whitwell, C. S. Woodwark, J. H. Wroughton, A. C. Young, C. L. C. Owen.

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Second Conjoint.—*Anatomy and Physiology.*—H. E. Ashley, F. M. Howell, J. W. Nunn, R. H. Paramore, A. T. Pridham, C. A. S. Ridout, P. H. Ross, E. Russell-Risien, E. G. Smith, A. E. Soden. R. L. Thornley, A. R. Tweedie, E. Wethered, R. H. R. Whitaker, M. G. Winder.

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Second Conjoint.—*Anatomy.*—W. M. James.

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University of Durham.—*Chemistry and Physics.*—G. Smith, P. E. Turner, B. E. G. Bailey, W. C. Douglas. *Anatomy, Physiology, and Materia Medica.*—H. G. Harris (*First in 2nd Class Honours*), R. Walker (*2nd Class Honours*), F. W. Crossman (*2nd Class Honours*), E. G. Klumpp, G. Smith, E. S. Wilkinson.

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Final L.R.C.P. and M.R.C.S.—The following have passed all parts of the final examination:—R. A. Fegan, G. D. Rowland, M. G. Dyson, J. H. Thursfield, A. W. Dickson, H. S. Beadles, H. Mundy, S. Hunt, R. G. Ralston, E. H. Betts, A. E. Ireland, J. Gundlach, J. E. Sandilands, H. C. Manning, E. J. E. Coop, A. Granville, P. Wood, B. E. Laurence, A. Woollcombe.

Obituary.

Hugh Robert Campbell Barber, B.A., M.B., B.C (Cantab.).

We have only just received notice of the death of Surgeon-Captain Barber, which took place, following enteric fever contracted in the discharge of his duties, at Murree, Punjab, India, on August 30th, 1896, at the age of 32. Though late, we hasten to chronicle this sad loss of an old Bart.'s man in the service of his country. Mr. Barber's ability may be gathered from the fact that he was a Scholar of St. Catherine's College, Cambridge, Gilchrist Scholar in the University of London, Clothworkers' Scholar, and Epsom Scholar of this Hospital. Subsequently to qualification he entered the Indian Medical Service, in which he was engaged at the time of his death.

Review.

Student's Note-book. Part I—Physiological Chemistry. By Arthur J. Hall, B.A., M.B.(Cantab.), M.R.C.P.(Lond.); Hon. Physician, Sheffield Royal Hospital; Lecturer on Physiology, School of Medicine. London: Baillière, Tindall, and Cox, 1897. 2s. 6d. net.

Of the importance of successful teaching in Physiological Chemistry there can be no doubt, for it is on chemistry that physiology must largely rely at the present moment for its further advance. And such an advance appears to be taking place along two lines—the synthesis of complex organic bodies, and the study of the micro-chemistry of the cell. The example of the first which will occur to all is Emil Fischer's work on the synthesis of the sugars. Few things have so cleared and simplified our views of a difficult problem. Even more suggestive are Dr. Pickering's as yet incomplete researches on the synthesis of colloid bodies, which may lead in time to the discovery of the structure of the proteid molecule. The other line of advance does not involve large questions such as these, but the mapping out of the various elements in the cell affords us aid by no means to be despised. For instance, McAllum's methods of detecting intra-cellular iron must help us to an under-

standing of some of the obscure points in the pathology of anæmia. It were easy to multiply examples if necessary, for biological problems probed deeply frequently become chemical problems, and it is to the chemist that we must turn for their solution.

It is because of the need and importance of a sound elementary knowledge of physiological chemistry that we welcome the little book before us. Dr. Hall is an old Bart.'s man, well known to many, who in his time played many parts in the Amateur Dramatic Club of the Hospital. His book is precisely what it aims to be, a practical guide for the student to have by him in the laboratory. It makes no attempt to replace text-books for reading, but is an admirable pilot among the shoals of chemical apparatus. By marginal notes the student is briefly reminded of the meaning and character of the tests he is doing, while the text is devoted to telling him precisely what to do. Any one who turns to Dr. Hall's instructions for using Fleischl's Hæmometer (p. 23) will bear out what we have said as to the eminently practical nature of the book.

Of the value of this schematic method of teaching physiology most teachers are now aware. It has been in use for a good many years at Cambridge, and Dr. Hall's plan does not differ widely from that of Dr. L. E. Shore's tables for elementary students, and Dr. Sheridan Lea's for advanced classes. But it most distinctly has its place between these two. Where the general execution is so good it may seem invidious to draw attention to weak points, but it does seem to us that the section on bile and that on urinary pigments are not equal in standard to the rest of the work.

It is well and clearly printed, and thoughtfully interleaved for note-taking. We have noticed but few typographical errors, though there is one unfortunate one on p. 18 (par. 2), where a "not" has crept in the text and spoilt the sense of the paragraph. But that can be altered in the next edition, which will doubtless soon be called for.

New Productions.

"SOLOIDS" OF COMPRESSED EUCAINE HYDROCHLORIDE (Burroughs, Wellcome & Co.).—Eucaine as a local anæsthetic has certain obvious advantages over cocaine. It is much less toxic, produces more extensive anæsthesia, keeps better, and can be sterilised by boiling without decomposition. Dr. W. J. Horne and Mr. Yearsley, in reporting thirty-six cases of its use at the Farringdon General Dispensary (*British Medical Journal*, January 16th, 1897), found that 5 per cent. or 8 per cent. solutions were efficacious in most cases, though a 2 per cent. solution was quite adequate for the uvula, laryngoscopy, posterior rhinoscopy, and aural examinations. Anæsthesia is slower in its onset with eucaine than cocaine, taking about ten minutes, and lasting ten to twenty minutes. In only three of their cases were any bad effects noted, and in each of these there were other conditions quite sufficient to account for the symptoms without referring them to the anæsthetics; further, it was successfully used where cocaine had produced bad effects. This drug has not been extensively employed in the throat department of this hospital, but the instances of its use confirm Dr. Horne's and Mr. Yearsley's observations. Its advantage for those patients who manifest an "idiosyncrasy" against cocaine is very considerable. But these authors mention two drawbacks which will prevent its replacing the better known drug entirely. It is apt to cause temporary salivation when applied to the mouth or fauces, thus hampering the operator; and it does not reduce the size of the turbinate bodies in the way that causes cocaine to be a valuable aid in diagnosis.

Of the efficacy of these convenient "soloids" of the drug recently prepared by Messrs. Burroughs, Wellcome & Co. we can speak from personal observation; one soloid dissolved in eleven minims of water makes a solution the strength of which is one in ten. But of their solubility we speak with more diffidence; it is by no means easy to dissolve one in eleven minims of water: we believe the manufacturers recommend the use of *warm* water, but this is not stated on the bottle.

"TABLOIDS" OF COMPRESSED RED GUM (Burroughs, Wellcome & Co.).—Here is an old friend in a new guise, and a pleasant guise too. By some recent improvements in the formula Messrs. Burroughs & Wellcome have produced a mild though efficient astringent for the throat, which they rightly claim does not "offend the palate or affect the appetite." These tabloids are supplied in bottles containing 100 in each.

Correspondence.

To the Editor of St. Bartholomew's Hospital Journal.

WOUNDS AND BRUISES IN THE INSANE.

SIR,—In connection with Dr. Claye Shaw's paper on "Wounds and Bruises in the Insane," the following case recently attended may perhaps seem of some interest.

A man aged about 53 had bronchitis, slight in itself, but attended with very violent cough. After a few days swellings appeared along the margins of the lower ribs in front. These turned out to be hæmatomata, the "bruising" and lividity from which extended over the whole abdominal region, and even on to the penis and scrotum. The hæmatomata were no doubt due to rupture of some fibres of the recti or of the lower intercostals; they were attended with severe pain. Beyond slight rhonchus there were no physical signs in the chest. The man was of neurotic temperament and very muscular. He certainly coughed with unnecessary violence at times.

This condition is, I suppose, a very unusual one, and had it occurred in an insane person in asylum practice it would have been hard to believe that he had not had a good pommelling from the attendants.

I am yours, &c.,
H. STANLEY.

Birth.

BENJAMIN.—On March 27th, at The Old Hall, Dorrington, Shrewsbury, the wife of J. K. Kinsman Benjamin, M.R.C.S., L.R.C.P., of a son.

Marriages.

PENNY—MACRAE.—On the 24th inst., at St. John's Church, Upper Norwood, by the Rev. H. S. Carpenter, Alfred Gervase Penny, M.A., M.B., B.C., of Peterhouse, Cambridge, and St. Bartholomew's Hospital, to Nellie Grace Gillanders, eldest daughter of the late John MacRae, of Arrowah, Bengal, and of Mrs. MacRae, of Ingleside, South Norwood.

SWABEY—STACE.—On the 22nd inst, at Widcombe Old Church, Bath, by the Rev. H. B. Swabey, father of the bridegroom, and the Rev. F. La Trobe Foster, vicar of Widcombe, Maurice Swabey, A.M.S., to Hilda Maunsell, daughter of Lieut.-Colonel E. V. Stace, C.B., Indian Staff Corps.

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